

Intestinal Ischemia After Laparoscopic Cholecystectomy

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ABSTRACT

Objectives: Intestinal ischemia is a rarely reported complication following laparoscopic cholecystectomy. We describe a case of massive small intestinal necrosis 3 days following this procedure. An autopsy determined the cause to be splanchnic hypoperfusion, likely due to the physiologic changes induced by the pneumoperitoneum necessary to conduct the surgery. We sought to determine whether all reported cases of intestinal ischemia following laparoscopic cholecystectomy (LC) shared the same etiology, and if faulty operative technique could be invoked.

Methods: A review of all known published cases of intestinal ischemia following LC was conducted to determine the underlying etiology of ischemia, and whether adequate operative technique had been observed.

Results: Multiple causes of intestinal ischemia following LC have been described: splanchnic hypoperfusion, thrombosis of the superior mesenteric artery, and thrombosis of the inferior mesenteric vein. All occurred despite correct operative technique.

Conclusions: Intestinal ischemia following LC has diverse causes. Patient factors rather than faulty operative technique would appear to underlie the development of this rare complication.

Key Words: Laparoscopic, Cholecystectomy, Intestinal, Ischemia.

INTRODUCTION

Intestinal ischemia following laparoscopic cholecystectomy (LC) is a rarely reported complication, but one that often results in a fatal outcome. Several case reports in the past decade have served to illustrate that ischemia in this context has diverse causes.¹⁻⁷ Most have been due to thrombosis of the superior mesenteric artery. In this report, we describe a case of fatal intestinal ischemia following LC of which splanchnic hypoperfusion during pneumoperitoneum was the most likely cause. We review the pathologic diversity of intestinal ischemia following LC.

CASE REPORT

A 57-year-old woman was referred for LC following a several month history of biliary colic. She was moderately obese but otherwise in good health, without any history or known risk factors for cardiovascular disease. LC was performed without complication according to the standard technique with maintenance of the intraperitoneal pressure between 12 mm Hg to 15 mm Hg. The patient was discharged home the same day with a prescription for analgesics to be taken as necessary. Shortly afterwards, she began to experience epigastric pain that responded well to analgesics. However, at home on the third post-operative day, she suddenly vomited a large volume of blood and died several minutes later in front of her husband. The coroner was consulted, and an autopsy performed at his request.

At autopsy, the operative site was unremarkable. However, the entire small intestine was green and obviously necrotic; the lumen contained a large amount of blood, as did the stomach. Microscopic examination confirmed the presence of full thickness necrosis of the small bowel wall. The mesenteric vessels were meticulously examined: these were normal without evidence of atheromata, arterial thromboembolia, or venous thrombus. The aorta was free of plaque. The heart was normal except for mild atheromatosis of the left descending coronary artery; the other coronary vessels were normal. The other organs showed no abnormality. The cause of death was recorded as massive small bowel ischemic necrosis due to arterial

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hypoperfusion, the latter most likely due to elevated intraabdominal pressure during the laparoscopic cholecystectomy procedure.

DISCUSSION

Intestinal ischemia following LC is a rare but devastating complication. A literature review using PubMed and pertinent search terms revealed 7 cases before the present report (**Table 1**). All but 2 patients died as a consequence. The causes of ischemia in the context of LC are diverse, and likely depend on the underlying cardiovascular health of the affected patient.

LC requires the creation of a pneumoperitoneum via insufflation of carbon dioxide; resulting increases in pCO₂ and intraperitoneal pressure lead to changes in pulmonary function and hemodynamic measurements.⁸ Pulmonary function is altered as a consequence of increased pCO₂ that is directly absorbed into the circulation from the peritoneal cavity as well as by increased intraperitoneal volume: vital capacity is diminished with increased dead space and V/Q mismatch. Increased intraabdominal pressure leads to decreased blood flow through the splanchnic vessels with relative hypoperfusion of the celiac, superior mesenteric, and renal arteries, despite normal blood pressure.⁹ Although these physiologic changes create a theoretical risk of compromised intestinal blood flow in all

patients, in the vast majority they are well tolerated with no clinical consequences.

The patient we describe had no clinical or autopsy evidence of significant cardiovascular disease. The absence of atheromatosis of the major intraabdominal and mesenteric vessels, and the absence of venous thromboembolism, both excluded by detailed examination at the time of autopsy, eliminates these as causes of ischemia. Likewise, faulty operative technique or intestinal obstruction due to external compression or herniation may also be eliminated. We conclude that the massive small intestinal ischemia in this patient is therefore most likely splanchnic hypoperfusion occurring at the time of pneumoperitoneum, the physiologic basis of which is described above.

Splanchnic hypoperfusion is but one cause of intestinal ischemia following LC. As seen in the accompanying table, the most common cause in this small number of cases is thrombosis of the superior mesenteric artery (SMA). Patients with underlying atheromatous disease involving the abdominal vessels may be at risk for this complication.² However, an alternative explanation in this context is that symptomatic episodes of intestinal ischemia (mesenteric angina) before LC have been misinterpreted clinically as symptomatic cholelithiasis.⁷ The resulting laparoscopic intervention may have created a low blood perfusion state resulting in further intestinal ischemia. One

Table 1.
Reported Cases of Intestinal Ischemia After Laparoscopic Cholecystectomy

Case	Age/Sex	Duration (Minutes)	IAP (mmHg)*	Onset of Symptoms	Distribution of Ischemia	Vascular Thrombosis?*	Outcome
Paul (1994)	68/M	85	15	Day 4	Ileum and right colon	?	Death
Jaffe (1994)	76/F	70	?	Day 3	Entire small bowel and right colon	Yes: SMA	Death
Thiele (1994)	87/M	?	?	Day 4	?	Yes: SMA	Death
Dwerryhouse (1995)	36/M	50	15	30 hours	Ileum	No	Recovery
Schorr (1995)	62/F	40	?	Day 3	Small bowel and right colon	Yes: SMA	Death
Klugewitz (1998)	41/M	?	?	24 hours	Colon	Yes: IMV	Recovery
Andrei (1999)	72/F	50	15	Day 8	Entire small bowel	?	Death
Leduc (2005)	57/F	120	15	Day 3	Entire small bowel	No	Death

*IAP = intraabdominal pressure; SMA = superior mesenteric artery; IMV = inferior mesenteric vein.

patient died from thrombosis of the inferior mesenteric vein, the cause of which is unexplained, and 1 patient, similarly to our case, developed ischemia secondary to splanchnic hypoperfusion; he subsequently recovered. The specific cause of ischemia was not given for the remaining 2 cases. The 2 patients who survived were relatively young with limited bowel involved. Of note, in all cases, there was correct operative technique reported; where pCO₂ is specified, it was 15 mm Hg or lower.

The question arises whether patients at risk for developing intestinal ischemia during LC can be identified and, if so, can any preventive steps be taken? Given the very small number of cases in which this complication has occurred amid the large number of LCs performed, the negligible incidence of intestinal ischemia likely makes any attempt at screening of the average LC patient—even if reliable screening criteria could be identified—a futile endeavor. The patient we describe is illustrative: a previously healthy 57-year-old, moderately obese woman without any other known risk factors for cardiovascular disease would not be considered at increased risk for this complication. Similarly, most of the patients described in the small number of case reports had no identifiable risk factor. Thus, at best, an individualized approach is warranted: measures such as use of lower peritoneal pressure, if technically feasible, and assurance of proper hydration might be considered for those patients with precarious cardiovascular status or a prior history of ischemic enteritis or colitis.

CONCLUSION

Intestinal ischemia following LC is rare. Autopsy in the present case was essential to determine the cause of death. SMA thrombosis, inferior mesenteric vein thrombosis, and splanchnic hypoperfusion are all potential causes,

all occurring despite correct operative technique. Compromised cardiovascular function may play a role in the development of ischemia in certain patients. The possibility of survival would appear to correlate with patient age and extent of bowel involved. Preventative measures may be considered on an individualized basis for those patients with severe cardiovascular disease or a prior episode of intestinal ischemia.

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